

Mechanisms for Radiation Dose-Rate Sensitivity of Bipolar Transistors

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Abstract—Mechanisms for enhanced low-dose-rate sensitivity are described. In these mechanisms, bimolecular reactions dominate the kinetics at high dose rates thereby causing a sub-linear dependence on total dose, and this leads to a dose-rate dependence. These bimolecular mechanisms include electron-hole recombination, hydrogen recapture at hydrogen source sites, and hydrogen dimerization to form hydrogen molecules. The essence of each of these mechanisms is the dominance of the bimolecular reactions over the radiolysis reaction at high dose rates. However, at low dose rates, the radiolysis reaction dominates leading to a maximum effect of the radiation.

Index Terms—Bimolecular reaction, bipolar junction transistor, cracking, dimerization, dose rate, ELDRS, excess base current, hole, hydrogen, interface trap, kinetics, proton, radiation, recombination, silicon dioxide.

I. INTRODUCTION

IONIZING radiation generally degrades the performance of Si microelectronic devices by creating interface traps and oxide-trapped charge [1], [2]. The interface traps consist of Si atoms at the interface that failed to bond to the oxide [3]. Such traps act as recombination centers for electrons and holes, and this recombination leads to increased base current in bipolar transistors [2]. The oxide-trapped charge can shift the gate voltage of MOS devices negatively, and it can cause large increases in leakage current [2].

The mechanisms for creating radiation damage have been examined in numerous studies [2]. It is generally agreed that ionizing radiation releases hydrogen that reacts with the interface to create interface traps [4], [5]. The best-accepted model, the two-stage hydrogen model, explains the effects in terms of protons that react with the interface [6], [7]. In this

mechanism, radiation creates holes that react with hydrogen source sites to release protons. These protons then migrate to the interface where they react with and release hydrogen from previously passivated traps. This process creates interface traps.

In certain bipolar devices, the buildup of interface traps has been shown to depend on the dose rate of the radiation source [8], [9]. This leads to enhanced low-dose-rate sensitivity (ELDRS) in these bipolar technologies. Several models have been proposed to explain ELDRS [10]–[15]. The most widely accepted model is a space-charge model [10], [11], [14], [15]. In this model, at high dose rates, the space charge of trapped holes slows the migration of protons to the interface, and thus fewer interface traps are produced. However, at low dose rates the space charge is too small to have an effect. As a consequence, approximately twice as many interface traps are produced at low dose rates [15].

We have developed a theoretical model for ELDRS in terms of the bimolecular reactions describing the radiation effects in an oxide. In our approach three main types of reactions are considered; these are recombination of free electrons and trapped holes, retrapping of hydrogen, and dimerization of hydrogen. We focus on these previously neglected reactions because they can produce the nonlinear effects that may lead to a dose-rate dependence.

In this paper, we discuss the dose-rate dependence that follows from consideration of bimolecular reactions. Assuming one particular bimolecular reaction, hydrogen retrapping by the source sites that release hydrogen, the basic idea is that atomic hydrogen released by sources can be retrapped. This becomes increasingly important during irradiation because the density of empty source sites steadily increases. This retrapped hydrogen becomes unavailable for creation of interface traps. If the irradiation is performed at a sufficiently low dose rate, molecular hydrogen, if present at a sufficiently large density, replenishes the empty source sites and thereby prevents retrapping. In principle, all the hydrogen released at low dose rates is available for creating interface traps. However, as the dose rate increases, retrapping consumes a larger fraction of the atomic hydrogen.

We also discuss the other reactions and their dependence on experimental parameters. Finally, we assess ELDRS and discuss the application of the bimolecular mechanisms to understanding the data.

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II. THEORY

Our theory is a generalization of the two-stage hydrogen theory, and it attempts to explain the key radiation effects phenomena in silicon dioxide following exposure to ionized radiation. We focus on interface trap creation due to release of hydrogen by electrons and holes created by the ionizing radiation. We assume that both neutral and ionic species of hydrogen can be released from defect sites that act as sources of hydrogen, ionic or neutral. We also consider the deposition of positive charge in the oxide. Our approach is to develop a computational model whose foundation is the well-known two-step hydrogen radiolysis model [6]. To do this, we define the minimum set of reactions that forms a computable model and estimate the parameters governing these reactions. To estimate parameters, we use electronic structure calculations based on density functional theory [16] together with empirical information and simple approximations based on insight.

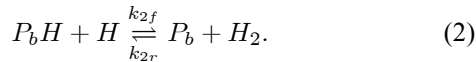
We begin by describing the key reactions, and we use these first in a discussion of the two-step hydrogen model and then in a discussion of a prototypical bimolecular reaction model that produces a dose-rate dependence. For both models we present a simplified discussion that focuses on the reaction kinetics and neglects explicit discussion of electric field driven transport within the oxide. In other words, we assume that diffusion is rapid compared with electric-field driven drift. One justification for this approximation is the absence of externally applied electric fields during irradiation in the ELDRS experiments.

A. The Key Reactions

The interface traps are assumed to be P_b -centers, the well-known defects at the Si-SiO₂ interface. These defects are the dangling bonds of Si atoms that failed to bond with O atoms in the oxide. These dangling bonds can trap carriers that then recombine with carriers of the opposite sign. Annealing of P_b -centers in a hydrogen environment causes hydrogen to bond to these Si atoms. The resultant P_bH defects are thereby passivated because they cannot trap carriers. However, atomic hydrogen can react with the passivated defect thereby recreating the original P_b defect. Studies have shown strong evidence that these phenomena are governed by the following exothermic reactions [17]:



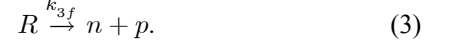
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These reactions are valid for either neutral hydrogen or protons; the charge is balanced by electron transfer at the interface. The practical result for the semiconductor industry is an annealing procedure that reduces the trap density $[P_b]$ to acceptable levels. The ultimate effect of the ionizing radiation, as envisioned in the hydrogen model, is to drive reaction (2) forward. This “depassivation” process produces interface traps, the P_b -centers.

At this point, we consider mechanisms for radiolytic effects due to hydrogen. In the two-step hydrogen model, the

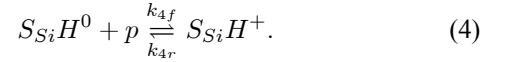
hydrogen is released by carriers created by ionizing radiation and then it migrates to the interface where it depassivates the P_b -center [6]. The radiolytic effects of hydrogen follow after electrons n and holes p are created by ionizing radiation; this process can be written as a reaction in which R represents radiation:



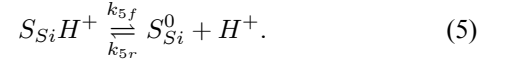
The first stage is the release of hydrogen from a site within the bulk of the oxide.

Once released, the second-stage in the model is the migration of the hydrogen to the interface and the depassivation of the P_b -centers [6]. Experiments using electrical bias during the transport stage have led to the conclusion that the transporting hydrogen species is the proton, H^+ [6], [18].

The two-stage hydrogen model assumes that ionizing radiation causes the release of protons. The radiation effects literature does not contain convincing evidence about the source of protons. However, electronic structure calculations suggest that hole capture stimulates the release of protons from $S_{Si}H$ (silanol) sites [19]–[21]; this result is consistent with experimental work on the two-stage hydrogen model [22]. In this mechanism, the trapping of a hole weakens the bond enough that the proton is released. First, a hole is captured:



Next, the resultant trapped species $S_{Si}H^+$ dissociates by releasing a proton, H^+ :



B. The Two-Stage Hydrogen Mechanism

We begin with a discussion of the well-known two-stage hydrogen mechanism. In this section, we present a simplified version of this mechanism whose predictions can be easily computed.

In this mechanism, the first step is the creation of electrons and holes by the ionizing radiation. We assume ionizing radiation at a constant dose rate g in $\text{rd}(\text{SiO}_2)$ per second. For a given total dose N_0 , the duration t_g is defined by the relation

$$N_0 = gt_g. \quad (6)$$

We assume electron-hole pairs are generated at a rate κg in which $\kappa = 8.1 \times 10^{12} \text{cm}^{-3} \text{rd}(\text{SiO}_2)^{-1}$ [23], and in this approximate treatment we assume that each pair creates a free electron and a hole.

In this mechanism, ionizing radiation stimulates the release of protons, a phenomenon represented by (3), (4) and (5). Using these reactions, we write kinetic equations governing the populations of the various species (for each species, we denote its concentration using brackets $[\]$; however, following tradition, we do not use this designation for electrons n and holes p). From these reactions, we obtain the following kinetic equation for the holes,

$$\frac{dp}{dt} = \kappa g - k_{4f}[S_{Si}H^0]p - r_p p, \quad (7)$$

in which the term $r_p p$ represents transport and other recombination of the holes. We also obtain kinetic equations for the other species:

$$\frac{d[S_{Si}H^+]}{dt} = k_{4f}[S_{Si}H^0]p - k_{5f}[S_{Si}H^+] + k_{5r}[S_{Si}^0][H^+] \quad (8)$$

$$\frac{d[H^+]}{dt} = k_{5f}[S_{Si}H^+] - k_{5r}[S_{Si}^0][H^+] - k_{2f}[P_bH^0][H^+] - r_{H^+}[H^+]. \quad (9)$$

Note that we have also included a term $r_{H^+}[H^+]$ for migration and other reactions of the protons. Finally, the interface trap density is governed by

$$\frac{d[P_b]}{dt} = k_{2f}[P_bH^0][H^+]. \quad (10)$$

In this approximation, we ignore the reverse reaction that becomes important at high temperature and high proton density. The areal interface trap density N_{it} is given by the following relation:

$$N_{it} = d[P_b], \quad (11)$$

in which d is the thickness of the oxide.

We assume each of these reactions reaches a steady state condition. For the holes, we obtain

$$p = \frac{\kappa g}{k_{4f}[S_{Si}H^0] + r_p}. \quad (12)$$

From the other two equations, we also obtain the steady state solution for the protons:

$$[H^+] = \frac{k_{4f}[S_{Si}H^0]p}{k_{2f}[P_bH^0] + r_{H^+}}. \quad (13)$$

This becomes

$$[H^+] = \frac{\varepsilon_p \kappa g}{k_{2f}[P_bH^0] + r_{H^+}}, \quad (14)$$

in which an efficiency for holes has been defined:

$$\varepsilon_p = \frac{k_{4f}[SH^0]}{k_{4f}[SH^0] + r_p}. \quad (15)$$

Using this solution,

$$[P_b] = \frac{k_{2f}[P_bH^0]\varepsilon_p \kappa g t_g}{k_{2f}[P_bH^0] + r_{H^+}}. \quad (16)$$

Then using $N_0 = g t_g$ and the efficiency for protons,

$$\varepsilon_{H^+} = \frac{k_{2f}[P_bH^0]}{k_{2f}[P_bH^0] + r_{H^+}}, \quad (17)$$

we obtain the final result

$$N_{it} = \varepsilon d \kappa N_0, \quad (18)$$

in which $\varepsilon = \varepsilon_p \varepsilon_{H^+}$ is a combined efficiency. As expected, this result is independent of dose rate.

C. Bimolecular Mechanisms and Dose-Rate Dependence

Each of the bimolecular mechanisms leads to a sublinear dependence on the total dose, and this leads to a dose rate dependence. This sublinear dependence will be illustrated by doing a simplified heuristic calculation. The more detailed and rigorous calculations are presented in the Appendix.

The sublinear behavior can be illustrated by considering one of the bimolecular mechanisms, the recombination of electrons and holes to form excitons X :

$$n + p \xrightarrow{k_{19f}} X. \quad (19)$$

In this case, we are considering the competition of bimolecular electron-hole recombination with the reaction that causes the release of protons as discussed above. The results reveal that this bimolecular recombination becomes dominant if the total dose and the dose rate are sufficiently large.

For this simplified calculation, we consider the following kinetic equations for the electrons and holes that are being generated at a rate κg :

$$\frac{dp}{dt} = \kappa g - knp - r_p p \quad (20)$$

$$\frac{dn}{dt} = \kappa g - knp - r_n n \quad (21)$$

with $k = k_{19f}$. In these equations, the transport terms are not explicitly considered. Instead, transport out of the sample is included in the terms r_p and r_n . Because electrons migrate much more rapidly than holes, $r_p \ll r_n$. Furthermore, we consider the irradiation durations short enough that hole migration can be neglected, and thus use the approximation $r_p = 0$. Given these conditions, the electron density reaches a steady state value but the hole density grows. Using the steady state condition for the electrons,

$$\kappa g - knp - r_n n = 0, \quad (22)$$

the kinetic equation for holes becomes

$$\frac{dp}{dt} = r_n n. \quad (23)$$

The kinetics of this equation depends on the dose rate g . In the limit of low dose rates, $r_n n = \kappa g$, and this equation predicts a linear buildup of the holes. In the limit of high dose rates, $knp = \kappa g$ because the bimolecular term governs the steady state concentration of electrons. For this case,

$$\frac{dp}{dt} = \frac{r_n \kappa g}{kp}. \quad (24)$$

After integration of this equation with the assumption that the initial hole concentration is zero, we obtain

$$p(t_g) = \sqrt{\frac{2r_n \kappa g t_g}{k}} = \sqrt{\frac{2r_n \kappa N_0}{k}} \quad (25)$$

using $N_0 = g t_g$, the definition for the total dose. This sublinear dependence on total dose at high dose rates leads to the dose rate dependence that is the focus of this paper.

Fig. 1 shows the total dose dependence of the low and high dose rate solutions. For the low dose rate case, the interface trap density grows linearly with total dose, the result

obtained from the two-stage hydrogen model. For this case, $N_{it} = \varepsilon d\kappa N_0$ in which $\varepsilon = \varepsilon_p \varepsilon_{H^+}$. For these calculations, we assume the conversion efficiency is one, thus each electron-hole pair releases one hydrogen atom and it creates an interface trap. Thus, at the lowest dose rate, each electron-hole pair causes the creation of an interface trap, a worst-case outcome that is admittedly physically unrealistic but necessary for this general analysis. Furthermore, the interface trap density does not saturate in the simple model because (1) has been omitted and the passivated trap density is held constant. Thus, at high total doses, the interface trap density can exceed the passivated trap density.

In the high-dose-rate limit, the dependence on dose becomes sublinear at high total dose, in agreement with (25). In this case, the dependence on dose is linear until the critical total dose N_c is reached. As discussed in the Appendix, this critical total dose is defined by the trap density r_f/k for which the hydrogen retrapping rate is equal to the hydrogen reaction rate with the interface. Above this critical dose, the trap density grows sublinearly with total dose, $N_{it} \propto N_0^{1/2}$.

Fig. 2 shows the interface trap density dependence on dose rate. This dependence is discussed in the Appendix for the hydrogen trapping mechanism. At very low dose rates, the interface trap density is constant at $\varepsilon d\kappa N_0$ until a dose rate $g_{c1} = r_s N_c$ is reached. Above this first crossover rate, the interface trap density drops as $N_{it} \propto g^{-1/2}$ until a dose rate $g_{c2} \simeq r_s N_0$ is reached. It then remains approximately constant at $\varepsilon d\kappa \sqrt{N_c N_0}$ as the dose rate increases. Although not shown in the figures, at very high dose rates the interface trap density drops to an asymptotic value $\varepsilon d\kappa N_c$. We note that there is no dose rate effect if $N_0 \lesssim N_c$.

D. Specific Bimolecular Mechanisms

There are other bimolecular mechanisms that produce a dose rate dependence that is similar to the one discussed for the electron-hole recombination mechanism. Each of them is briefly discussed in this section.

There are at least three variants to the electron-hole recombination mechanism. One, which involves free electron and free hole recombination, has been described already. Another involves free electrons recombining with trapped holes. In this variant of the mechanism, the holes are captured at traps that release these holes slowly. The primary reaction involves hole capture at trap sites:



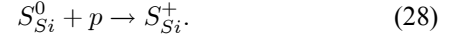
The other reaction involves electron capture that neutralizes these trapped holes:



The ELDRS effect arises due to the competition between these two reactions. At high dose rates, the second reaction dominates, and this reduces the density of holes available to release protons from other sites.

Another electron-hole recombination mechanism involves a trapped hole that releases hydrogen directly by "cracking" a

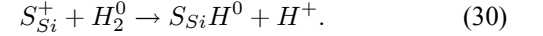
hydrogen molecule to release a proton. In this mechanism, holes are captured at hydrogen source sites:



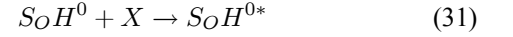
The ELDRS effect for this case arises from the competition between the electron neutralization reaction



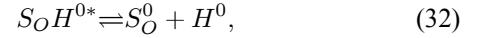
and the hydrogen cracking reaction



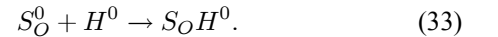
Another bimolecular mechanism involves hydrogen retrapping at source sites (S_O) that released the hydrogen. This mechanism is proposed because experiments have also shown that radiolysis can also release neutral hydrogen that can be observed using electron spin resonance (ESR) measurements [24]. The neutral hydrogen is likely released from non-bridging oxygen defects. Two different mechanisms have been suggested, either electron or exciton capture [25]. Assuming exciton capture, the postulated kinetic equations are:



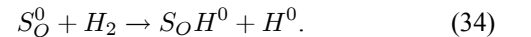
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in which S_OH^{0*} is an activated species that may release the hydrogen atom before cooling. The equations are similar if electron capture stimulates the hydrogen release. Subsequently, the cooled species S_O^0 may retrap the hydrogen:



We also assume that the hydrogen at the source sites can be replenished if the concentration of molecular hydrogen H_2 is sufficiently high:



In agreement with previous work, we assume that the released hydrogen can react with passivated interface traps to create interface traps. The dose rate dependence follows from the competition between the retrapping reaction and the cracking reaction. The solutions for this mechanism are described in the Appendix.

Finally, the hydrogen dimerization mechanism involves a dimerization reaction of two neutral hydrogen atoms to form a hydrogen molecule:



This is a well-known reaction [26]. The dose rate dependence follows because, at high dose rates, this reaction competes with (2), the hydrogen depassivation reaction.

III. OVERVIEW OF THE EFFECTS OF BIMOLECULAR MECHANISMS

In this section, the common features and differences between the mechanisms will be summarized. As stated at the outset, each mechanism has been treated in the same level of approximation to facilitate comparisons. For example, transport of migrating species such as electrons, holes and protons has been described in terms of an extra recombination rate, and this rate has led to an efficiency factor for each mechanism. This efficiency factor also depends on the electric field but the explicit effects of electric fields have also been ignored.

Given these approximations, each of the bimolecular mechanisms leads to qualitatively similar dose rate dependences but each depends on different physical parameters. As expected, the conventional two-stage hydrogen mechanism has no dose rate dependence:

$$N_{it} = \varepsilon d \kappa N_0. \quad (36)$$

Furthermore, each of the bimolecular mechanisms reduces to an expression of this form in the low dose limit. As the dose rate increases, the end result is that each bimolecular mechanism can be described by three characteristic rates. These are a slow rate r_s , a fast rate r_f , and a bimolecular rate k . The combination of these quantities leads to a critical total dose

$$N_c = \frac{r_f}{\varepsilon \kappa k} \quad (37)$$

and a critical dose rate

$$g_c = \frac{r_s r_f}{\varepsilon \kappa k} = r_s N_c. \quad (38)$$

Each of these characteristic quantities must be exceeded in order for ELDRS effects to be observable.

The effect of transport is explicitly included in the complete model but not in this simplified discussion of the essence of our model. An external electric field tends to increase the parameters N_c and g_c ; similarly, reducing the oxide thickness has the same effect. These effects occur because the carriers spend less time in the oxide. However, inclusion of the internal electric field can have the opposite effect because the internal electric field of the slowly migrating holes tends to retain the electrons in the oxide. Thus, inclusion of the internal field enhances electron-hole recombination (non-geminate). Therefore, the parameters N_c and g_c are reduced, leading to more pronounced ELDRS effects. Furthermore, the recombination of electrons with trapped holes is expected to reduce the effects of the space charge mechanism [15].

The observance of ELDRS in a particular material technology depends on the material properties of the particular oxides under consideration. These properties determine which mechanism dominates and also the critical quantities for that mechanism. Generic estimates for these quantities using the concept of diffusion-limited reactions are shown in Table I. Our estimates for these parameters lead us to suggest that critical dose rates ranging from 10^{-8} to 10^2 rd(SiO₂)/s can be expected from these bimolecular mechanisms. Finally, we observe that these mechanisms can produce time dependent effects with a time scale determined by $1/r_s$. The

table suggests time delays of approximately 10^6 s may occur for the slowest mechanisms.

IV. APPLICATION OF THE MECHANISMS

In this section, we begin the comparison with data by examining the qualitative features of the mechanisms. This qualitative approach is necessary because there are no cases for which enough data have been obtained for a base oxide to define the parameters of any of the mechanisms. This is especially true because a base oxide is often very defective due to processing that takes place after it is grown. Also, this oxide is usually encapsulated by another deposited layer, a passivation layer, to reduce the effects of contaminants.

Ignoring the details of the base oxide, a major qualitative prediction of the theory is that the true radiation sensitivity of microelectronic devices is only revealed at low dose rates. As the dose rate is increased, the various bimolecular recombination mechanisms become increasingly important, and the effect of these mechanisms is to obscure the true radiation sensitivity. Furthermore, each mechanism tends to lessen the radiation effect. Thus at higher dose rates the mechanisms described in this report tend to mitigate the effects of ionizing radiation, and this leads to an apparent improvement in radiation hardness at high dose rates. Accordingly, instead of ELDRS, one might use the term, "Reduced High-Dose-Rate Sensitivity".

Another general prediction is a sublinear dependence on the total irradiation dose. This can be seen in the high dose rate limit shown in Fig. 1. Similar sublinear effects have been observed in several studies [27]. To explain these results, we adopt the following generic approach because of lack of information and insight about oxide layers in these earlier studies. Whatever the release mechanism for hydrogen, either electrons or holes must be involved. Furthermore, we assume the oxide samples have electron and hole traps that may have reached high concentrations. If we make this assumption, the electron-hole recombination mechanism may explain the sublinear total dose dependence observed in these experiments.

A further prediction is the dose-rate dependence, the motivation for this work. Fig. 3 compares the predicted interface trap density as a function of dose rate with experimental data. These data show the dependence of the change in base current for a series of transistor irradiations at various dose rates [28]. One can observe that the theory is in good qualitative agreement with the data. This qualitative agreement is expected because the excess base current is proportional to the interface trap density [29].

Another general prediction is the presence of time-delayed effects. These are present in each of the electron-hole recombination mechanisms. These effects lead to a delayed response to irradiation, and these effects have been observed experimentally. In particular, one time-delayed effect, latent interface trap buildup (LITB) occurs on a 10^6 sec time, and it can be explained as a delayed effect due to hole traps that release hydrogen by a cracking reaction [30], [31].

Recent experiments have shown that the type of passivation layer is important in determining whether ELDRS does or does not occur [31], [32]. This was demonstrated by observing

that ELDRS was eliminated by removing the silicon nitride passivation from various integrated circuits [32]. Furthermore, ELDRS was absent in new devices prepared with no passivation layers, but a variety of radiation effects occurred in devices made with new passivation layers [32]. In summary, the earlier work showed that passivation was implicated in causing ELDRS but none of the substitute passivation layers eliminated the problem.

In future work this theory will be applied to these recent experiments on the effects of passivation layers [32]. These studies have concluded that either the hydrogen source sites or mechanical stress due to the passivation layer controls whether ELDRS does or does not occur [32]. Assuming our theory has merit, we assume that the type of passivation layer governs the critical quantities, g_c and N_c , and thereby determines whether ELDRS is observed.

A major practical task is to measure the true radiation sensitivity of devices in spite of the obscuring effects of ELDRS. This theory, if validated, can help accomplish this task because it predicts that the radiation response is linear at very low dose rates, the worst case situation. Furthermore, the response remains linear, independent of dose rate, if the total dose is sufficiently small. This can be seen in Fig. 1 in the limit that the total dose is lower than the characteristic dose. This straightforward situation is complicated by the fact that the theory also predicts that the radiation effects are time-delayed for various mechanisms. Combining these ideas suggests that one can perform a sequence of time-dependent experiments to measure the true radiation response of devices.

V. CONCLUSION

In conclusion, we have described a theory for the dose-rate dependence of the effects of ionizing radiation on interface traps in microelectronic oxides. In this theory, bimolecular reactions that consume radiolytic species lead to ELDRS. The approximate calculations based on this theory are consistent with earlier experiments that have shown a sublinear dependence on total dose and with ELDRS experiments. Furthermore, this theory can be used to explain the dependence of ELDRS on the types of passivation layer used. However, further work is needed to determine if the theory can predict the dose-rate dependence in terms of measureable parameters that describe the passivation layers. Finally, the theory can be used to define a worst-case test of the radiation hardness of microelectronic devices.

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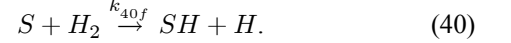
APPENDIX

To illustrate the calculations to be described, we analyze the solutions for the hydrogen capture mechanism. In our simplified treatment of the theory we focus on the essential reactions. We ignore the distinction between forms of atomic

hydrogen, neutral or ionic, because one can envision similar mechanisms for both forms of hydrogen. We assume hydrogen migration is rapid compared with the duration of the experiments to be explained. We assume that ionizing radiation releases hydrogen from the source sites at a rate $\varepsilon\kappa g$ in which ε is the efficiency, and this hydrogen can be retrapped by the source sites S :



We also assume that the hydrogen at the source sites can be replenished if the concentration of molecular hydrogen H_2 is sufficiently high:



In agreement with previous work, we assume that the released hydrogen can react with passivated interface traps to create interface traps.

We obtain the following kinetic equations for the empty traps and the hydrogen:

$$\frac{d[S]}{dt} = \varepsilon\kappa g - k[S][H] - r_s[S] \quad (41)$$

$$\frac{d[H]}{dt} = \varepsilon\kappa g - k[S][H] - r_f[H]. \quad (42)$$

In these equations $k = k_{39f}$, $r_s = k_{40f}[H_2]$, and $r_f = k_{2f}[P_bH]$. Finally, the interface trap density is governed by

$$\frac{d[P_b]}{dt} = r_f[H]. \quad (43)$$

These equations will be solved approximately to develop insight. As will be further discussed, the two populations evolve in distinct stages, and approximate solutions can be constructed for each of these stages.

The various time stages are the following. Initially, the hydrogen and empty source densities are both increasing. First, the hydrogen density reaches a quasi-steady-state condition while the empty-source density continues to grow. If the dose rate is high enough, bimolecular recombination then begins to dominate. Whether bimolecular recombination does or does not dominate, both species reach a steady state at long times. Finally, both populations decay after the irradiation ceases.

In the approximate solution, the transient time stages are ignored. The transient buildup of the hydrogen density, prior to reaching its steady state solution, is ignored, and the transient decay at the end of the irradiation is also ignored. Both are important if the irradiation duration is shorter than the bimolecular recombination time. Both effects will be ignored in the analytic solutions but they are included in the numerical calculations.

The solution we describe is valid at all dose rates except for very large dose rates. It is a sum of the contributions from two time stages. In the first stage, the hydrogen and empty trap populations are growing, and in the second stage, the hydrogen and empty trap populations are assumed to have reached constant steady state values. The transition from time-varying to steady state solutions occurs at a time t_c that is defined by the condition that the time-varying solutions have reached the steady state values.

In the first stage, the solution at short times, both populations are increasing. However, the replenishment rate due to hydrogen cracking is small, and thus it can be neglected. Then the equations become

$$\frac{d[S]}{dt} = \varepsilon\kappa g - k[S][H] \quad (44)$$

$$\frac{d[H]}{dt} = \varepsilon\kappa g - k[S][H] - r_f[H]. \quad (45)$$

By inspection of these equations, both densities will grow at the same rate at first. Eventually, the last term in (45) will limit the growth of hydrogen leading to the quasi-steady-state condition

$$\varepsilon\kappa g - k[S][H] - r_f[H] = 0. \quad (46)$$

Solving for the quasi-steady-state hydrogen density yields

$$[H(t)] = \frac{\varepsilon\kappa g}{k[S(t)] + r_f}. \quad (47)$$

If the bimolecular term begins to dominate, the hydrogen density decays as the empty trap density continues to grow. Both populations will evolve until either the irradiation has ended at time t_g or until they have reached steady-state values at time t_{ss} . Given these conditions, $t_c = \min(t_g, t_{ss})$.

Using (46) in (44), produces the differential equation

$$\begin{aligned} \frac{d[S(t)]}{dt} &= r_f[H] \\ &= \frac{\varepsilon\kappa g r_f}{k[S(t)] + r_f}. \end{aligned} \quad (48)$$

Solving this equation, assuming that $[S(0)] = 0$, produces the result:

$$[S(t)]^2 + \frac{2r_f}{k}[S(t)] = \frac{2\varepsilon\kappa g r_f t}{k}. \quad (49)$$

This yields the following:

$$[S(t)] = -\frac{r_f}{k} + \frac{r_f}{k} \sqrt{1 + \frac{2\varepsilon\kappa g k t}{r_f}}. \quad (50)$$

The interface trap density $[P_b(t)]$ can be obtained by solving

$$\frac{d[P_b(t)]}{dt} = r_f[H(t)], \quad (51)$$

an equation identical to (48). Thus, using the solution (50),

$$[P_b(t)] = \varepsilon\kappa N_c \left(\sqrt{1 + \frac{2gt}{N_c}} - 1 \right) \quad (52)$$

in terms of the critical total dose

$$N_c = \frac{r_f}{\varepsilon\kappa k}. \quad (53)$$

In the second time stage, both populations have reached steady state, constant values. For this case,

$$\varepsilon\kappa g - k[S_{ss}][H_{ss}] - r_s[S_{ss}] = 0 \quad (54)$$

$$\varepsilon\kappa g - k[S_{ss}][H_{ss}] - r_f[H_{ss}] = 0. \quad (55)$$

These equations require that the condition

$$r_s[S_{ss}] = r_f[H_{ss}] \quad (56)$$

must be satisfied. The above equations lead to the following quadratic equation for the hydrogen density:

$$\frac{kr_f}{r_s}[H_{ss}]^2 + r_f[H_{ss}] - \varepsilon\kappa g = 0. \quad (57)$$

Solving this equation,

$$[H_{ss}] = \frac{r_s}{2k} \left(-1 + \sqrt{1 + \frac{4k\varepsilon\kappa g}{r_s r_f}} \right). \quad (58)$$

At the lowest dose rates, the first term in (57) can be ignored, and thus

$$[H_{ss}] \approx \frac{\varepsilon\kappa g}{r_f}. \quad (59)$$

Substituting in (43),

$$[P_b(t_g)] = \varepsilon\kappa N_0, \quad (60)$$

a result that is independent of dose rate.

At higher dose rates the bimolecular term dominates, and thus

$$\frac{kr_f[H_{ss}]^2}{r_s} \approx \varepsilon\kappa g. \quad (61)$$

For this case,

$$\begin{aligned} [P_b(t_g)] &= r_f[H_{ss}]t_g \\ &= r_f \sqrt{\frac{\varepsilon\kappa r_s g}{kr_f}} t_g \\ &= \sqrt{\frac{\varepsilon\kappa r_s r_f}{kg}} N_0 \end{aligned} \quad (62)$$

using $N_0 = gt_g$. By inspection, this leads to a dose rate dependence.

Having the two solutions, the steady state time t_{ss} is obtained by matching the time evolving solution to the steady state solution, the condition that $[H(t_{ss})] = [H_{ss}]$. Thus, using (47) and (58),

$$\frac{\varepsilon\kappa g}{r_f \sqrt{1 + \frac{2k\varepsilon\kappa g t_{ss}}{r_f}}} = \frac{r_s}{2k} \left(-1 + \sqrt{1 + \frac{4k\varepsilon\kappa g}{r_s r_f}} \right). \quad (63)$$

In the low dose rate limit, $[H_{ss}] = \frac{\varepsilon\kappa g}{r_f}$. By inspection, $t_{ss} = 0$ in this limit.

The complete solution is a sum of the growth and steady state terms:

$$N_{it}(t_g) = d\varepsilon\kappa N_c \left(\sqrt{1 + \frac{2gt_c}{N_c}} - 1 \right) + dr_f[H_{ss}](t_g - t_c). \quad (64)$$

In this expression, we have used $N_{it} = d[P_b]$ and

$$t_c = \min(t_g, t_{ss}). \quad (65)$$

Note that t_{ss} is obtained from (63) with the condition that $t_{ss} > 0$.

Now we can examine the solutions as a function of dose rate. At very low dose rates, $t_c = 0$, and then the interface trap density is independent of dose rate. As the dose rate increases, the trap density falls, and the second term becomes less important. At high dose rates, the solution is given by the first term alone. For this case, $t_c = t_g$, and thus

$$N_{it} = d\varepsilon\kappa N_c \left(\sqrt{1 + \frac{2N_0}{N_c}} - 1 \right) \quad (66)$$

at high dose rates. Furthermore, if $N_0 \ll N_c$ then $N_{it} \simeq d\epsilon\kappa N_0$, and if $N_0 \gg N_c$ then $N_{it} \simeq d\epsilon\kappa\sqrt{2N_0N_c}$.

The two crossover dose rates for the transition from a constant to a dose-rate dependent solution can be estimated by finding the dose rates at which the dose-rate dependent solution equals the constant solutions. The first crossover dose rate g_{c1} follows from the condition that the linear and quadratic terms in (54) are equal. This is achieved for a dose rate

$$g_{c1} = \frac{r_s r_f}{\epsilon\kappa k} = r_s N_c. \quad (67)$$

Now the second crossover dose rate g_{c2} can be estimated. Once again this is done by equating two expressions. Equating (62) to N_{it}/d obtained from (66) produces the expression

$$\sqrt{\frac{r_s r_f}{\epsilon\kappa k g_{c2}}} N_0 = \sqrt{\frac{2r_f N_0}{\epsilon\kappa k}}, \quad (68)$$

and thus

$$g_{c2} = \frac{r_s N_0}{2} \simeq r_s N_0. \quad (69)$$

At the very highest dose rates, one can assume all the hydrogen is produced instantly. Thus the initial concentrations are $[H(0)] \approx \epsilon\kappa N_0$ and $[S(0)] \approx \epsilon\kappa N_0$. In a simple analysis one can reason that all the hydrogen and traps react until the bimolecular term no longer dominates. This occurs when $[H] \approx \epsilon\kappa N_c$. This remaining concentration of hydrogen is then fully consumed in producing the interface traps. Thus

$$[P_b] \approx \epsilon\kappa N_c \quad (70)$$

in this limit.

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TABLE I

ESTIMATES OF BIMOLECULAR PARAMETERS

Mechanism	$r_s (s^{-1})$	$r_f (s^{-1})$	$k (cm^3 s^{-1})$	$g_c (rds^{-1})$	$N_c (rd)$
Hydrogen dimerization	1	1	10^{-15}	10^2	10^2
Hydrogen retrapping	10^{-4}	1	10^{-15}	10^{-2}	10^2
Slow hydrogen release	10^{-6}	10^4	10^{-7}	10^{-8}	10^{-2}
Slow hole release	10^{-2}	10^4	10^{-7}	10^{-4}	10^{-2}
Hydrogen cracking	10^{-6}	10^4	10^{-7}	10^{-8}	10^{-2}

FIGURES

Fig. 1. Dependence of interface trap density on total dose for the two limiting cases, low dose rate and high dose rate. The sublinear growth occurs if the total dose exceeds a minimum total dose N_c .

Fig. 2. Dependence of interface trap density on dose rate. At low dose rates, the interface trap density saturates to $\varepsilon\kappa dN_0$, in which N_0 is the total dose, and, at high dose rates, it saturates to $\varepsilon\kappa d\sqrt{N_0N_c}$.

Fig. 3. Dependence of transistor base current on dose rate. The theoretical results of this work are in qualitative agreement with previously published data.

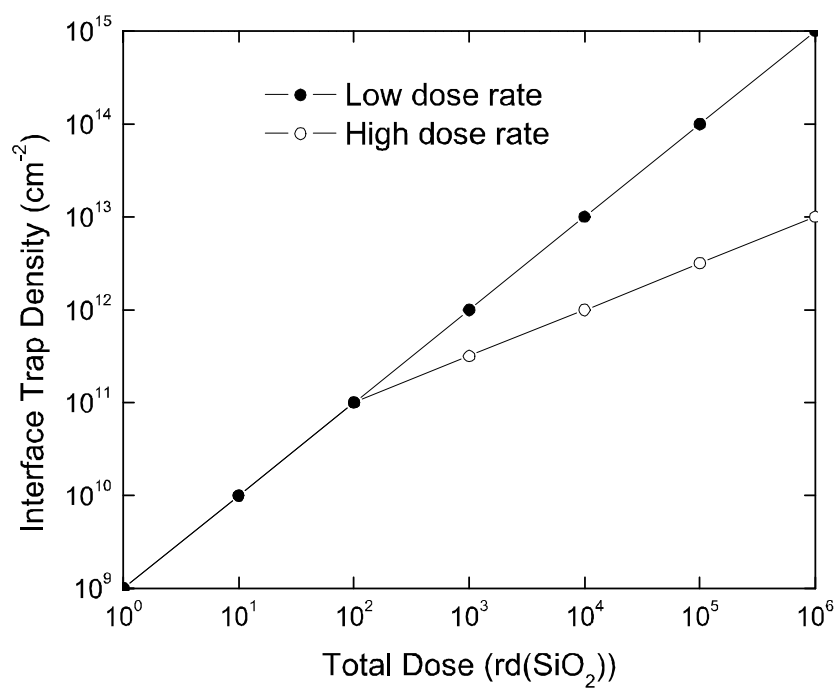


Fig. 1.

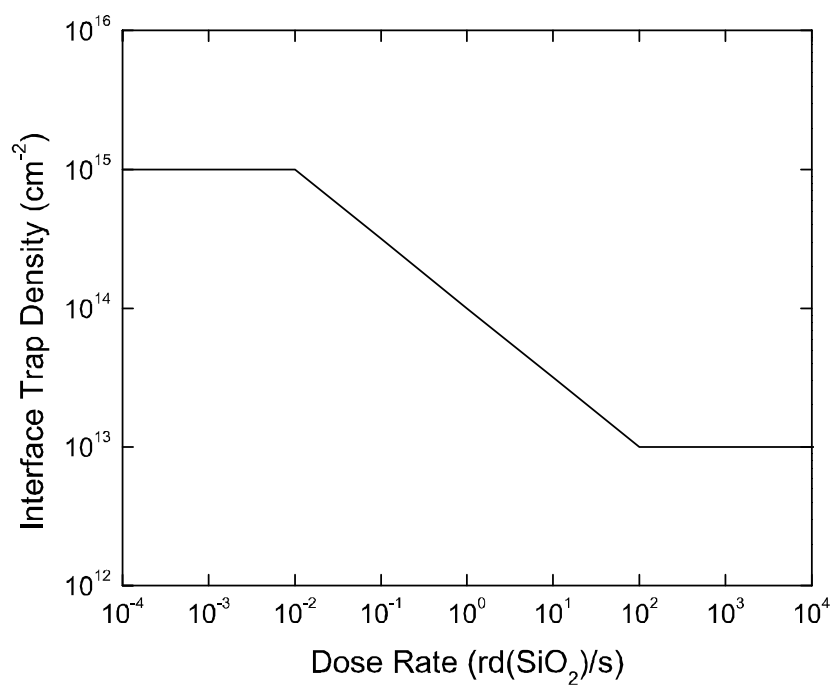


Fig. 2.

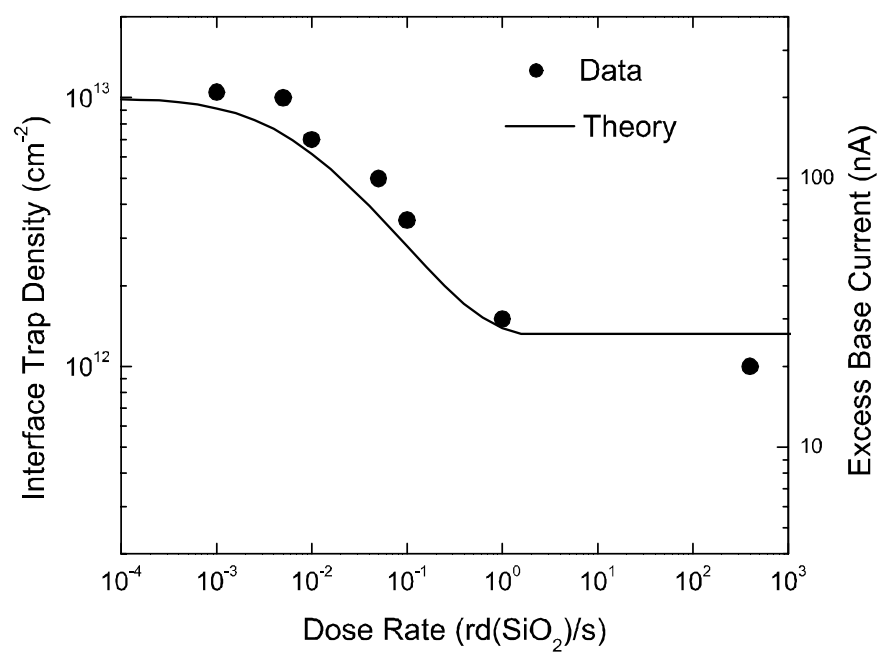


Fig. 3.